A successful renal transplantation in Behçet’s syndrome

Renal involvement is not frequent in Behçet’s syndrome (BS) and consists of occasional reports of patients suffering from glomerulonephritis,1 IgA nephropathy2 and renal amyloidosis.3 We present the successful outcome of a renal transplantation in a patient who had end stage renal failure secondary to glomerulonephritis. To our knowledge, this is the first patient with BS to receive an organ transplantation.

The detailed history of this patient at the time of the diagnosis of glomerulonephritis was the subject of a case report in 1991.1 In brief, she was 21 years old when she developed recurrent oral and genital ulcers, bilateral uveitis, erythema nodosum, folliculitis, and intermittent arthritis of the knees. Two years later, she was referred to our centre for further evaluation of eye symptoms. She had no active mucocutaneous lesions at that time, the pathergy reaction was positive and cerebrospinal fluid analysis was normal. It was decided to prescribe only local drops for her mild eye involvement. Three months later she experienced two ocular episodes resulting in a sharp decline of visual acuity and azathioprine 2.5 mg/kg/day was prescribed. Two weeks later she was admitted to the hospital because of microscopic haematuria. She was ANA negative, the anti-DNA and serum complement levels were normal. Her glomerular filtration rate was 67 ml/min. An open renal biopsy showed diffuse proliferative glomerulonephritis and weak focal segmental positivity of IgA and IgM. She was treated with three boluses of 1 g methylprednisolone and was discharged prescribed azathioprine 150 mg/day, aspirin 300 mg/day and prednisone 30 mg/day. She was well except for occasional mucocutaneous symptoms and a mild intermittent ocular episode during the next four years. However her renal function deteriorated progressively despite uninterrupted treatment with azathioprine and changing doses of prednisone and she was put on regular haemodialysis. The graft function was good. A week in the 14th month of haemodialysis, she received a kidney from her mother. The graft function started immediately and she was prescribed maintenance immunosuppression with azathioprine, cyclosporin A and methylprednisolone. An acute interstitial type rejection on the 11th day of transplantation was treated successfully with pulsed corticosteroids. Now 40 months after transplantation, she has normal renal function and is free of any symptoms of BS except for occasional oral ulcers.

We had some hesitation in performing a renal transplantation in our patient initially because of the lack of any previous experience and particularly because of our concern for the heightened inflammatory response of BS patients to simple penetrating trauma that is best characterised by the pathergy reaction.4 This reaction, however, is not only limited to the skin and development of aneurysms after vascular punctures and episodes of synovitis after arthrocentesis have been observed.5 Furthermore, postoperative complications leading to a poor outcome such as occlusions of grafts/anastomoses after the surgical treatment of aneurysms6 or paravalvular leakage and suture breakdown after aortic valve replacement7 have been reported in BS patients. As these complications are probably related to the pathergy phenomenon of BS, you would also reasonably expect problems after an organ transplantation, an operation with arterial and venous anastomoses. On the other hand, we had previously shown that despite the increased inflammation, wound healing after full thickness skin punch biopsies is not changed in BS.8 We have not experienced any of the feared complications after the transplantation procedure in the last 14 months. Whatever it might be related to, the outcome in our patient suggests that BS patients can undergo renal transplantation with a satisfactory outcome.

SUHEYLA APAYDIN
EGEN ULUC
Division of Nephrology, Department of Internal Medicine

VEDAT HAMURYUDAN
HASAN YAZICI
Division of Rheumatology, Department of Internal Medicine

MUZAFFER SARIYAR
Department of Surgery, Cerrahpasa Medical Faculty, University of Istanbul, Istanbul, Turkey

Correspondence to: Dr V Hamuryudan, Vesipsapa sokak 100, Yil Sitesi, I Blok D16 Uskudar, 81190 Istanbul, Turkey.


Data were compared for significance for Student’s unpaired t test.

Table 1 summarises lymphocyte phenotypes in patients with SSc and healthy controls.

We found a higher expression of T cell activation marker CD25+ and NK cell major surface marker CD56+. In lymphocyte phenotypes, there was not any difference among disease subsets and CD25+ and CD56+ were not correlated with the disease duration.

Immune system abnormalities have been suspected in the development of SSc because of the presence of autoantibodies, altered cytokine production and evidence of overlap with other autoimmune diseases. It was suggested that immune system changes play the major part in the development of vasculopathy and fibrosis. Previous reports on T lymphocyte subpopulations in SSc are partially conflicting. Melendro et al9 demonstrated that there was no significant difference in the levels of CD4+ and CD8+ T cells in 22 SSc patients and control group but in rheumatoid arthritis (RA) CD3+ and CD8+, in Sjögren’s syndrome CD3+, CD4+ and CD8+ levels were significantly decreased compared with those of controls and they suggested that the abnormalities in immune regulatory T cell circuits leading to autoimmunity are different in each connective tissue disease.

Table 1 Lymphocyte phenotypes in patients with SSc and healthy controls

Serum Systemic sclerosis (n=29) Control group (n=12) t Test p Value

CD3 (%) 71 (9) 69 (9) 0.660 >0.05
CD4 (%) 44 (9) 45 (9) 0.110 >0.05
CD8 (%) 31 (9) 29 (8) 1.914 >0.05
CD4/CD8 3.56 (0.6) 1.84 (0.6) 1.339 >0.05
CD19 (%) 12 (3) 13 (5) 0.445 >0.05
CD25 (%) 18 (9) 7 (3.5) 4.150 <0.05
CD56 (%) 22 (9) 14 (5) 2.691 >0.05

**Unpaired Student’s t test. Data shown as mean (SD).

Lymphocyte phenotypes in systemic sclerosis

Although the pathophysiology of systemic sclerosis (SSc) is not fully clarified, there are considerable data implicating abnormalities of microvascular changes, fibroblast activation and immune system abnormalities. Immune system abnormalities are reported as a stimulus in both fibrotic and vascular damage.1 To investigate the immune system abnormalities in the pathogenesis of SSc we evaluated lymphocyte phenotypes in patients with SSc and healthy controls. Immune system abnormalities are probably leading to a poor outcome such as occlusions of grafts/anastomoses after the surgical treatment of aneurysms or paravalvular leakage and suture breakdown after aortic valve replacement have been reported in BS patients. As these complications are probably related to the pathergy phenomenon of BS, you would also reasonably expect problems after an organ transplantation, an operation with arterial and venous anastomoses. On the other hand, we had previously shown that despite the increased inflammation, wound healing after full thickness skin punch biopsies is not changed in BS. We have not experienced any of the feared complications after the transplantation procedure in this instance. One possible reason for this favourable outcome might be that our patient was female. It is known that BS runs a milder disease course in women compared with men.2 Additionally, the rather intensive immunosuppressive/anti-inflammatory post-transplant drug use might also have contributed to the diminished disease activity of our patient as well as to the prevention of a reaction at the site of transplantation. Whatever it might be related to, the outcome in our patient suggests that BS patients can undergo renal transplantation with a satisfactory outcome.

Lymphocyte populations and cytokine concentrations in peri-cardial fluid from a systemic lupus erythematosus patient with cardiac tamponade

Pericardial involvement is the most common cardiovascular complication in systemic lupus erythematosus (SLE). The clinical picture varies from subclinical pericardial effusion and classic acute pericarditis to cardiac tamponade. Several studies of pericardial fluid (PF) have been limited to determination of autoantibodies, complements and immune complexes. To further study the pathogenic mechanisms involved in lupus pericarditis, we measured the lymphocytic populations and cytokine concentration pattern in PF and peripheral blood (PB) from a SLE patient with cardiac tamponade.

We report a case of a 38 year old man with SLE diagnosed in December 1995 when he presented with polyarthralgias, photosensitivity, oral ulcers, nephritis, non-hemolytic anemia, positive ANA, increased anti-dsDNA and hyocomplementemia. The patient improved with corticosteroid and intravenous cyclophosphamide treatment. However, on 18 June 1997 he presented with syncope, hypotension (80/40 mm Hg), a tachycardia, jugular vein distension and cardiomegaly. The two dimensional echocardiogram showed a large pericardial effusion with right atria and ventricle collapse in diastole. Pericardial tap was performed and 180 ml of an orange fluid was aspirated. Examination of PF showed white blood cell count of 5280/mm³ (polymorphonuclear cells = 96%). The absolute number of lymphocytes was lower in PF than in PB (211 ± 700/mm³). PF concentration was much higher in PF than in PB. After a 2 month follow up, he remained clinically stable without recurrent pericardial involvement or SLE exacerbations.

Table 1 Frequency of lymphocyte populations and cytokine concentrations in peripheral blood and pericardial fluid

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Peripheral blood</th>
<th>Pericardial fluid</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lymphocyte population (%)</td>
<td>T cells 57.8</td>
<td>50.0</td>
</tr>
<tr>
<td></td>
<td>CD4+ T cells 17.6</td>
<td>25.0</td>
</tr>
<tr>
<td></td>
<td>CD8+ T cells 34.3</td>
<td>25.0</td>
</tr>
<tr>
<td></td>
<td>B cells 7.8</td>
<td>8.3</td>
</tr>
<tr>
<td></td>
<td>NK cells 34.3</td>
<td>41.7</td>
</tr>
<tr>
<td>Cytokine concentration (pg/ml)</td>
<td>IL1β 3.0</td>
<td>240.0</td>
</tr>
<tr>
<td></td>
<td>IL6 24.3</td>
<td>3.8</td>
</tr>
<tr>
<td></td>
<td>IL8 &lt;6.0</td>
<td>&lt;6.0</td>
</tr>
<tr>
<td></td>
<td>IL4 &lt;6.0</td>
<td>&lt;6.0</td>
</tr>
<tr>
<td></td>
<td>TNFα 3.8</td>
<td>15.4</td>
</tr>
<tr>
<td></td>
<td>INFγ 1.5</td>
<td>32.8</td>
</tr>
</tbody>
</table>

*Manufacturer (Genzyme, Boston, MA) detection limits: 3 pg/ml for IL1β, INFγ and TNFα; 4 pg/ml for IL2; 6 pg/ml for IL6; 8 pg/ml for IL8; and 5 pg/ml for IL10.

Level of protein was 4.1 g/dl (serum = 5.3 g/dl), glucose was 53 mg/dl (serum = 110 mg/dl) and LDH was 471 IU/l (serum = 110 IU/l). PF cultures were negative. No malignant cells were seen to possess markers for acute leukemias or lymphomas. PF contained high dose corticosteroids and azathioprine. Prednisone was gradually decreased to 10 mg daily over a month period. After a 2 month follow up, he remained clinically stable without recurrent pericardial involvement or SLE exacerbations.


Feriha Budak GÜHER GÖRAL Uludag University, School of Medicine, Department of Infectious Disease Correspondence to: Dr K Dilek, Uludag University Medical Faculty, Department of Nephrology and Rheumatology 16059, Gönülle-Bursa, Turkey.
tissue are necessary as the composition of lymphocyte and cytokine profiles may differ between pericardial fluid and tissue.

Luis M Vilà, Department of Internal Medicine, Universidad Central del Caribe School of Medicine, Bayamón, Puerto Rico

José R Rivera del Río

Department of Internal Medicine, Universidad Central del Caribe School of Medicine, Bayamón, Puerto Rico

Luis M Vilà, Department of Internal Medicine, División de Reumatología, Universidad Central del Caribe School of Medicine, Bayamón, Puerto Rico

Correspondence to: Dr. L. M. Vilà, Department of Internal Medicine, Division of Rheumatology, Universidad Central del Caribe School of Medicine, Call Box 60327, Bayamón, Puerto Rico 00960–6032, USA.

MATTERS ARISING

**RS3PE: six years later**

We read with interest the paper by Cantini et al and would like to comment on it.1

In 1992 we performed a retrospective multicentre study of 276 RS3PE patients. We concluded that personal pathogenetic link between both forms of AU. It is possible that the long term follow up of these patients will clarify whether or not it is the same entity.

Now, six years later, we have reviewed the original cohort of patients with the RS3PE syndrome. A questionnaire was sent to the participating rheumatologists. The survey focused on articular symptoms, treatment and evolution. The current cohort was composed of 22 patients (male 16; female 6; mean age:77.9; range 64–91). Four patients died (the three with haematological diseases, one stroke) and one was not located. Thirteen patients were asymptomatic and without treatment, in contrast nine required treatment, namely corticosteroids (6), gold salts (1), cloroquine (1) and NSAID (1). Interestingly, two of the patients were identified by their rheumatologist as having a seronegative rheumatoid arthritis, another patient had a chronic disease with separate corticosteroid responsive episodes of bilateral hand oedema and polyosymatic syns at different times. Last but not one patient developed Raymond’s phenomena, both hands had scolocytic response. A nailfold capillary microscopy showed a decreased number of capillary loops, which were widened, suggesting systemic sclerosis.

Our results suggests that RS3PE syndrome has a good prognosis and half of the patients are asymptomatic and without treatment six years later. However, there is a subset of patients that have other diseases. Although pure RS3PE syndrome does exist the evolution should be closely monitored.

**Authors’ reply**

We appreciate the comment by Olivé et al on our article on RS3PE. They reviewed 27 previously described RS3PE patients after a follow up of six years. As we suggested in a previous report, they confirm that RS3PE syndrome should be considered a heterogeneous condition associated with different inflammatory rheumatic diseases and also with neoplastic disorders.

In our study none of the 23 patients with RS3PE syndrome developed remissions supporting the diagnosis for another disease. The different study design and selection of patients may in part explain the subset of patients with other diseases and with a worse prognosis observed by Olivé et al.

We designed a prospective follow up study excluding patients satisfying the criteria for the diagnosis of polyalgia rheumatica, rheumatoid arthritis and seronegative spondyloarthropathies. Moreover, patients with a clinical history of cancer were excluded from the study. In their original report these authors performed a retrospective study including all patients with remitting distal extremity swelling with pitting oedema. They recruited also patients not evaluated for spondylarthropathies, which may be associated with distal extremity swelling with pitting oedema.1

However, in their retrospective evaluation Olivé et al found that 13 of 22 (59%) patients were asymptomatic and drug free over a six year follow up period, confirming that RS3PE not associated with other conditions and with a good prognosis does exist.

The problem is how to label this clinical picture. As discussed in our article, the similarities of demographic, clinical and MRI findings between patients with “pure” RS3PE syndrome and those with polyalgia rheumatica and the concurrence of the two syndromes suggest that these conditions may be part of the clinical spectrum of the same disease. In the series of Olivé et al the patient with a clinical course characterised by alternate relapses of HLA-B27 associated oedema or polyalgia symptoms further supports our hypothesis. Even those RS3PE patients successively diagnosed as having seronegative rheumatoid arthritis (elderly onset rheumatoid arthritis) do not conflict with our conclusions. Healey described patients who developed episodes of polyalgia rheumatica and seronegative rheumatoid arthritis at different times during follow up.2

Similar clinical characteristics have been recently described in a population based cohort of patients with giant cell arteritis followed up over a 42 year period. Four of the six patients who fulfilled the criteria for the diagnosis of rheumatoid arthritis during the follow up experienced multiple separate episodes of symmetrical arthritis, proximal symptoms of polyalgia rheumatica and distal extremity swelling with pitting oedema.2

**Letters, Matters arising**

**FRANCISCO RIVERA-CIVICO**

**JUAN JIMÉNEZ-ALONSO**

**MARIA MARTÍN-ARMADA**

**MARIA TERESA HERRANZ**

**JOSE CASTRO**

**FRANCISCO PEREZ-ALVAREZ**

**J. L. DEL ARBOL**

Service of Internal Medicine

**MANUEL TORIBIO**

Service of Ophthalmology

**FRANCISCO SAMANIEGO**

Service of Clinical Chemistry and Immunology,

“Virgen de las Nieves” University Hospital,

Granada, Spain

Correspondence to: Dr J Jiménez-Alonso, Jefe del Servicio de Medicina Interna, Hospital General de Especialidades “Virgen de las Nieves” Avda Fuerzas Armadas 2, 18014 Granada, Spain.


9 Rothova A, van Veenendaal WG, Linssen A, Glauser E, et al. Raynaud’s phenomena, both hands had sclerocytic response. A nailfold capillary microscopy showed a decreased number of capillary loops, which were widened, suggesting systemic sclerosis.


11 Correspondence to: Dr J Jiménez-Alonso, Jefe del Servicio de Medicina Interna, Hospital General de Especialidades “Virgen de las Nieves” Avda Fuerzas Armadas 2, 18014 Granada, Spain.

Correspondence to: Dr Cantini, 2nd Divisione di Medicina, Ospedale di Prato, Piazza Ospedaled 1, 59100 Prato, Italy.

1 Olivieri I, Salvarani C, Cantini F. Remitting seronegative symmetrical synovitis with pitting oedema (RS3PE) syndrome: a prospective fol-


4 Pollock JP, Roughley P, DiBattista J, McColl-

rum R, Martel-Pelletier J. Are cytokines in-


19.


Author’s reply

We agree with all the points made by Dr McCarthy. Basic calcium phosphates (BCPs) in synovial fluids may be important, and it may be that their identification will be validated in relation to future treatments. However, she seems to agree with the only two points made about BCPs in our article (which is about the identification of urate and pyrophosphate crystals): that is, that on the basis of current understanding BCPs are of “doubtful significance”, and that their identifi-

cation should have no influence on contem-

porary therapeutic decisions.

PAUL DIEPPE

MRC Health Services Research Collaboration, University of Bristol, Bristol

Mortality in rheumatoid arthritis patients

The paper “Mortality in rheumatoid arthritis patients with disease onset in the 1980s” is of considerable interest. A decrease in mortal-

ity risk for rheumatoid arthritis (RA) patients in more recent years would be important, but only in the first 10 years of RA. How-

ever, this inception cohort differs from those previously published so that no direct compa-

rison is possible. As earlier (and older and larger) studies have shown standardised mor-

tality ratios of two to three, a finding of “nor-

mal” mortality might imply that more recently used treatment strategies are revers-

ing the excessive mortality in RA previously described.

Yet, even at first perusal, there are a lot of deaths in this series of relatively young people. In the 10 years after a mean age of 51, 18 patients (10%) had died. Over 20 deaths were said to be “expected”. However, using US mortality rates for a population mean aged the same, projected over 10 years, two thirds women, and white, one would expect only 11 deaths using 1996 mortality rates, and deaths in 1985 rates, over the 1710 patients years of follow up. While we did not have the age distribution of this RA cohort to calculate precise expectations, these figures should be conservative. Female mor-

tality rates in the US white female popula-


3 Halverson PB, McCarty DJ. Patterns of radio-


4 Carroll GJ, Stuart RA, Armstrong JA, Breidahl PD, Laing BA. Hydroxyapatite crystals are a frequent finding in osteoarthritic synovial fluid, but are not related to increased concentrations of keratan sulphate or interleukin 1b. J Rheuma-


5 Schumacher HR. Synovial inflammation, crys-


6 McCarthy GM, Mitchell PG, Struve JS, Che-


9 Nair D, Misra RP, Sallis JD, Cheung HS. Phos-

phoro-pyrophosphate inhibits a basic calcium phospho-

tided in the same way that monosodium urate (MSU) or crystal pyrophosphate dihydrate (CPPD) can be by polarised light microscopy.

Furthermore, the presence of apatite crystals does not change the management of either osteoarthritis (OA) or any other arthropathy in patients at present. Doherty and Swan conclude therefore that apatite crystals are irrelevant to clinical practice. Historically, the role of cytokines in the pathogenesis of OA was also considered to be speculative. As with apatite crystals, levels of IL1, TNF-alpha, etc interleukin 1 (IL1) or tumour necrosis factor a (TNF) are not routinely measured in joint fluid from patients with arthritis. After considerable further investigation, however, the roles of IL1 and TNF in articular joint destruction and cartilage degeneration in OA are now considered important. As a consequence of such recognition, Pelletier and coworkers have prevented the development of OA in an experimental model by transfer of the IL1 receptor antagonist gene. We have shown that apatite crystals induce MMP-1 in human OA (HOA) fibroblasts with a potency equivalent to that of IL1 and TNF in vitro. Furthermore, apatite crystals induce IL1 and TNF-alpha act in synergy to increase MMP-1 production by HOA fibroblasts. Efforts continue to discover methods to inhibit the pathogenetic effects of IL1 and TNF. Why not inhibit the effects of apatite crystals also? Currently, there is no drug available to retard the progression of OA. A greater understanding of the pathogenesis of OA is essential to the development of rational treat-

ment thus allowing us to target important pathogenic mediators. While it might be tempting to write apatite crystals off as new age nonsense, a considerable body of evi-

dence suggests, like cytokines, they could serve as a novel therapeutic target as well as a prognostic marker. Without further study, only those with crystal balls can tell.

GERALDINE M MCCARTHY

Department of Clinical Pharmacology,

Royal College of Surgeons in Ireland and Mater Misericordiae Hospital, Dublin


2 Halverson PB, McCarty DJ. Patterns of radio-


3 Halverson PB, McCarty DJ. Patterns of radio-


4 Halverson PB, McCarty DJ. Patterns of radio-


6 McCarthy GM, Mitchell PG, Struve JS, Che-


tion, at 3.4 per 1000 per year at age 51 and 9.0 per 1000 per year at age 61 are presumably higher than those in long lived Malmöhus County, Sweden.

Of interest, in our own larger study with meticulously computed “expected” values in four different populations we also had an “expected” death rate of about 10% to 15% over 10 years. But, these were not inception cohorts and their age at start of follow up was 60.4, 62.6, 59.8, and 69.1 years. Thus, they were much older cohorts. Given the expected doubling of mortality rates each eight years (Gompertz’s law), expected deaths should have been two to three times more in our cohorts than in a cohort beginning at age 51.

Finally, recent studies have not suggested that “rheumatoid” deaths in themselves are the cause of the increased mortality in RA. The observed “excess” deaths are spread around in multiple disease categories, with accelerated atherosclerosis numerically the largest problem and only a slight relative increase in systemic RA complications, gastrointestinal haemorrhage, and infections.

Authors’ reply

We were pleased to notice the interest in our paper shown by Drs Fries and Bloch. In reply to their comments we do not consider the death rate of 10% in the cohort as an excessive one compared with the age and sex matched general population. It is not possible to calculate more precise figures of expected deaths knowing the mean age of the cohort only. To clarify this and make comparison possible we enclose a table of the age distribution in our cohort in five year intervals giving the number of observed and expected deaths for each age interval separately.

Women do live longer in Malmöhus County, Sweden than in the US. Female mortality rates in Malmöhus County were 3.76 per 1000 at age 51 and 7.32 per 1000 at age 61 in 1985. In 1996 the corresponding figures were 2.03 per 1000 at age 51 and 3.39 per 1000 at age 61.

We agree that the main cause of death in RA patients very seldom is the rheumatoid disease in itself. This was true also for our study where no certain connection between RA and death was found in any of the cases.

JAMES F FRIES
DANIEL A BLOCH
Stanford University, School of Medicine, Palo Alto, California, USA


Authors’ reply

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ELISABET LINDQVIST
KERSTIN EBERHARDT
Department of Rheumatology, Lund University Hospital, Sweden

Table 1

<table>
<thead>
<tr>
<th>Age group (years)</th>
<th>Number of patients</th>
<th>Expected mortality</th>
<th>Observed mortality</th>
</tr>
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<tbody>
<tr>
<td>15-19</td>
<td>1</td>
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<tr>
<td>20-24</td>
<td>3</td>
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<td>25-29</td>
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<td>30-34</td>
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<td>2</td>
</tr>
<tr>
<td>All</td>
<td>183</td>
<td>20</td>
<td>18</td>
</tr>
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</table>

Crystals in arthritis: new age nonsense or novel therapeutic target?

GERALDINE M MCCARTHY

Ann Rheum Dis 1999 58: 723
doi: 10.1136/ard.58.11.723

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